Running Phthalates to Ground

Pinpointing Exposure Sources in a Virtual Home

Methods to measure concentrations of chemicals in adults and children, a science known as biomonitoring, can be costly and burdensome. And, although biomonitoring data provide useful aggregate information on exposure to all sources, it is almost impossible to tell how much comes from a specific source. A new mechanistic model may offer a way to identify the strongest sources of exposure to semivolatile organic compounds by showing how chemicals move from a single product through a home and which model parameters have the greatest influence on exposure [***EHP* 118:253–258; Xu et al.**].

Researchers created a model of a hypothetical three-room house equipped with adjustable airflow systems to illustrate how human exposure to phthalates released by a specific source—in this case, vinyl flooring—might be predicted. Phthalates are plasticizers that are used in products as diverse as nail polish, plastic wiring, and children's toys. Data from the Centers for Disease Control and Prevention suggest that more than three-quarters of the U.S. population may be exposed to these suspected endocrine disruptors.

The research team built on an earlier model that described how diethylhexyl phthalate (DEHP)—one of the most prevalent phthalates—is released from vinyl flooring into air and sorbs strongly to interior surfaces (walls, ceilings, floors, furniture, etc.) and suspended particles. Here the researchers used the model to explore the relative importance of inhalation of vapor, inhalation of particles, dermal sorption of DEHP, and oral ingestion of household dust on total exposure levels. To test which parameters might change the amount of total DEHP exposure through different routes, the researchers varied model parameters such as the amount of ventilation and velocity of air moving through their model house.

Exposure to gas-phase phthalates from vinyl flooring can occur through ingestion, inhalation, or dermal absorption.

For example, they calculated that a fan pushing air through the house would cause more skin contact with phthalates by increasing the release rate from the vinyl surfaces to the air. The fan also thinned the layer of air cushioning the skin, increasing the transfer of DEHP from air to skin. Stagnant air without the fan caused less transfer of DEHP from air to skin, thus protecting against dermal uptake of DEHP.

The new model suggests that levels of phthalates measured in adults and children may result in part from contact with surfaces that may absorb high concentrations of DEHP, such as clothing. Changing the variables in the model house—from airflow to the amount of DEHP in the vinyl flooring to square footage in a room—made a difference in estimated exposure levels. Varying the parameters in this simple model demonstrated the potential for DEHP exposures arising from a single product to differ by as much as 40 times from one situation to another. That variability underscores the wide range of possible exposures across the population—and the difficulty of relying on biomonitoring alone to identify the most harmful sources.

Naomi Lubick is a freelance science writer based in Zürich, Switzerland, and Folsom, California. She has written for *Environmental Science & Technology*, *Nature*, and *Earth*.

Traffic Marker?

Early Exposure to Air Pollution Associated with Childhood Asthma

Asthma is now the most common chronic disease for children and a major cause of emergency room visits, hospitalizations, and school absences, according to the World Health Organization. Now a large population-based study has shown an association between elevated exposure to air pollution *in utero* and during the first year of life and a higher risk of asthma in preschool-aged children [***EHP* 118:284–290; Clark et al.**].

There are multiple known risk factors for developing asthma, including genetic factors, diet, and exposure to secondhand tobacco smoke and allergens. The speed with which the disease has risen in most developed and developing countries suggests environmental exposures probably play a prominent role. Although air pollution is known to worsen existing asthma, a succession of recent studies is building evidence for an additional association between exposure to traffic-related air pollution and initial onset of asthma in children.

Using a nested case–control study design, researchers looked at administrative and health care data for nearly 3,500 children born in southwest British Columbia, Canada, in 1999 and 2000 who were diagnosed with asthma by age 4 years. Each case was age- and sex-matched to 5 randomly chosen controls born in the same region and time period.

To estimate air pollutant exposures, the researchers mapped the residential history of each child against air pollution data obtained from regulatory monitoring, land use regression modeling, and proximity to stationary pollution sources and to roads. These metrics were used to calculate average exposures for the duration of the mother's pregnancy and the child's first year of life. Nine pollutant exposures were evaluated: carbon monoxide, nitric oxide, nitrogen dioxide, particulate matter (PM_{10} and $PM_{2.5}$), ozone, sulfur dioxide, black carbon, and wood smoke.

The highest risk of asthma was associated with exposure to the traffic-related pollutants carbon monoxide, nitric oxide, nitrogen dioxide, and black carbon; lesser associations were seen with exposure to PM_{10} and sulfur dioxide, as well as with proximity to industrial point sources. Proximity to roads was not associated with increased risk, but only a small number of children resided near major roads in the study population. Associations between air pollution and asthma were generally greater in girls than in boys, although asthma was significantly more common in boys, consistent with other populations. The authors observe that other researchers also have reported stronger associations in girls, although the finding is not entirely consistent.

This is one of the few studies to examine the effect of *in utero* exposure on pediatric asthma risk. However, because of relatively high correlation between *in utero* and first-year exposures, the relative importance of these periods could not be discerned.

Laura Alderson has written for *EHP* since the 1990s. She is a freelance writer based in Raleigh, North Carolina, specializing in medicine, science, and high technology.